Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

January 3, 1905.

The President *pro tem.*, DR. J. ARTHUR BOOTH, in the Chair.

A Case of Graves' Disease in a Child.—This case was presented by Dr. J. Arthur Booth. The patient was a girl, 13 years old, whose family history was negative. Her parents were both healthy, and there was no history of any degenerative disease in either branch of the family. The child had suffered from measles, whooping cough and scarlet fever in infancy. She had never had a convulsion. There was no history of fright, nor could any other etiological factor be discovered.

Eighteen months ago, that is, when the child was 11 months old, the exophthalmus was first noticed. Six months later the enlargement of the thyroid became noticeable, and during the past year all her symptoms had become more aggravated. The exophthalmus and thyroid enlargement were very marked, as well as the tachycardia, the pulse ranging between 120 and 140 per minute. In addition to these symptoms there was tremor, general nervousness and insomnia, also occasional headaches.

Dr. Booth said the interesting feature of the case was the early age at which the disease had manifested itself. It was usually observed between the 15th and 25th or 30th year. There were four other children in this patient's family, who were all enjoying good health.

Adenoma of the Pineal Gland, Occluding the Aqueduct of Sylvius, with Escape of Cerebro-Spinal Fluid Through the Nose and Perforation of the Frontal Horn of the Right Lateral Ventricle.—This case was reported by Dr. Adolf Meyer. The patient was a male. In 1894, when diving, he struck the top of his head. Following this, he complained of headache. He became blind in 1898, with chiasma symptoms. In 1899 he had transitory attacks of numbness on the left side of the face, leaving out part of the area of the middle branch. From that time on there was difficulty in moving the lower jaw towards the left. There was no atrophy of the masseter. In August, 1900, cerebro-spinal fluid began to drip from the right nostril, with considerable relief of the general symptoms. On the few occasions when the flow stopped the patient would become sleepy and stuporous for two or three days. During May and October, 1902, the patient, who was then 22 years old, had several general convulsions. It was during that year that he was presented by Dr. Meyer at one of the meetings of the New York Neurological Society. In January, 1904, death occurred in a status epilepticus. The only permanent symptoms had been weakness of the left side of the jaw, a small area of loss of pain sensation of the left lower corner of the mouth, and subjective numbness in the left side of the tongue. The gait remained normal; the knee-jerks were both increased; there was no clonus.

Autopsy: A tumor was found in the form of an adenoma of the pineal gland. It had pressed itself through the roof of the mid-brain behind the posterior commissure, protruding into the third and fourth ventricle and displacing the posterior corpora quadrigemina; the adenoma had practically no sand, and but slight pigmentation. The right lateral ventricle had a funnel-shaped depression in its anterior end, with a perforation through the cortex. In the case reported by Wollenberg (*Arch. f. Psychiatrie und Nervenkrankheiten*, 31, p. 206) there was a tumor of the occipital lobes and perforation of both frontal lobes, but there was no occlusion of the ventricle to help account for the perforation.

In connection with the case reported by Dr. Meyer, photographs made by Dr. C. I. Lambert were presented, and Dr. C. B. Dunlap demonstrated some microscopic sections.

Dr. Joseph Fraenkel spoke of the rarety of these cases, and complimented Dr. Meyer upon his careful and thorough report of the case under discussion.

Dr. Meyer, in reply to a question, said the only motor symptoms the patient presented were those of the masticatory segment, which showed in the deviation of the jaw to the right whenever the mouth was opened. In other words, there was a weakness of the left pterygoid muscles. It was impossible to demonstrate any atrophy of the masseters. There were no motor symptoms referable to the upper extremities.

A Case of Old Fracture-Dislocation of the Spine, with Paralysis, Followed by Recovery; After an Interval of Four Years Gradual Development of a Spastic Paraplegia and Sensory Symptoms, Referable to the Same Level of the Cord.—This case was presented by Dr. J. Ramsay Hunt. The patient was a man 45 years old, a laborer. Twenty years ago he had a venereal sore, followed by a suppurating bubo. He received internal medication for one month. No secondary symptoms were noted. He has had numerous attacks of gonorrhea, followed by the development of strictures. At present a stricture of small calibre exists in the membranous portion of the urethra.

In 1895 he met with a severe accident, falling three stories through an airshaft. He was unconscious for a few minutes, and was taken to Bellevue Hospital in an ambulance. Following the accident he complained of severe pains in the back, and both legs were paralyzed, although not completely, as he could move them slightly in bed. It was necessary to catheterize him a few times after the injury, but this difficulty soon passed away. After the accident an angular deformity of the spine was noted, which still exists. It was located about the level of the twelfth dorsal vertebra, and it was safe to assume that the spine had suffered a fracture-dislocation at that point, and that the spinal cord had been injured at the same time.

After remaining in bed for two months the legs began to improve. This improvement continued, and six or seven months later he was able to resume his occupation as a truck-driver, a laborious one, which necessitated the lifting of heavy weights. One year after the injury he was able to do a full day's work, and was suffering no pain nor inconvenience. He admitted, however, that his legs were not quite as limber nor as strong as they were before the accident. He could not run as fast as formerly, owing to a slight stiffness in the knees. There were no sensory nor vesical symptoms during this period.

For four years or more the patient remained in this condition. He drove a truck, and led the vigorous outdoor life of a laboring man.

About three years ago there developed, very gradually, symptoms of trouble in the lower extremities. He began to complain of sensations of numbness and cold in the feet and legs, accompanied by stiffness and weakness. Upon stooping he felt a numbness in the lower portion of the back, extending down the posterior thighs and legs. During the past two years these symptoms had progressed slowly and steadily without any sudden exacerbation, and without acute pain. The left leg was weaker than the right, and it was important to note that this was true of the initial paralysis and the long period of disability during convalescence.

At the time of the report the patient's condition was as follows: He had a well-marked kyphosis, the tip of which corresponded to the twelfth dorsal vertebra. Corresponding to this there was a posterior bilateral band of hyperesthesia at the same level. The spinal column was fairly mobile, and was not tender to direct pressure or on jarring. There has been no change in the degree of the kyphosis since the accident. There was spastic paraplegia of the lower extremities, with the spasticity and clonus more marked on the left side. Babinski's phenomenon was present on both sides. The skin reflexes were present, and there were distinct objective sensory disturbances in the lower extremities, including touch, pain and temperature, more pronounced on the right side. The man was able to stand fairly well with his eyes closed. He had considerable difficulty in urination, with occasional incontinence. He also complained of sharp, painful sensations and prickling down the posterior aspect of the thighs and legs, and on stooping he felt numb from the hips down. His sexual power was apparently unaffected. Coarse myokymic twitchings were present in the back below the level of the lesion; also in the buttocks, thighs and legs, especially their posterior aspect, after exertion and exposure to the cold. Occasionally during the past year he had sharp, shooting pains in the course of the sciatic nerves, causing reflex movements in the legs; these were more pronounced on the left side.

A Case of Fracture-Dislocation of the Spine Causing a Unilateral or Partial Lesion of the Cord.—This case was presented by Dr. J. Ramsay Hunt. The patient was an elevator operator, 22 years old, who about two months ago fell six stories with his elevator. He was unconscious fifteen minutes after the fall. He was removed to the Gouverneur Hospital, where it was found that he had suffered a fracture-dislocation of the spine, the deformity corresponding to the eleventh and twelfth dorsal and the first and second lumbar vertebræ. In addition there was a fracture of the internal and external malleoli of the left ankle joint.

The case was seen by Dr. Hunt six weeks later through the courtesy of Dr. John Rogers. At that time there was an oval area of anesthesia to touch, pain and temperature over the anterior and lateral surfaces of the right thigh. The right knee-jerk was absent; the right Achilles jerk was present and not exaggerated. The left leg was weak and spastic, with clonus. The Babinski reflex was not elicitable on either side. A girdle sensation was felt in the lower abdominal region, especially on the right side. There was difficulty on urination. Dr. Hunt said the case was a good example of a unilateral or partial lesion of the cord, resulting from a fracture-dislocation of the vertebræ.

The interesting features of the first case were the recovery from the initial paralysis, the long interval of comparative normal function, during which period a laborious occupation was practiced, and then the gradual reappearance of cord symptoms referable to the same level of the cord. The progression of the disease had been most insidious, but always steadily advancing, and of such a nature as to suggest a very gradual compression of the spinal cord.

It seemed reasonable to infer in such a case that the cord was undergoing gradual compression in the spinal canal already narrowed by the fracture-dislocation, and produced by an osseous growth (chronic proliferating osteitis) plus pachymeningeal thickening and adhesions. Spinal syphilis or chronic inflammatory changes within the cord could not be excluded, but Dr. Hunt believed that surgical measures should be taken to relieve the compression.

Dr. Joseph Fraenkel said it was rather difficult to decide the exact nature of the lesion in Case I shown by Dr. Hunt. Pressure on the cord due to an osseous lesion, he thought, would give rise to more severe symptoms than were present in the case, and he suggested the possibility of a post-traumatic hematomyelia.

Dr. Booth agreed with Dr. Fraenkel that an exostosis would probably give rise to more pain.

Dr. Hunt, in closing, said that in studying the case the possibility of a post-traumatic syringomyelia had been considered, as well as syphilis, or a chronic inflammatory condition of the cord, originating at the site of the old injury. The theory that the symptoms followed a hematomyelia, he thought, could be discarded, from the fact that such a lesion was generally found in the gray matter of the cord, and that it would have given rise to dissociated sensory symptoms. In the first case shown it is true there were coarse myokymic twitchings, but no atrophies and no disassociated sensory symptoms. The symptoms were those of a gradual compression of the cord. In cases where the compression was of insidious onset, sharp pains were often absent. In the case shown there were pains of sufficient severity to suggest involvement of the posterior roots.

The differential diagnosis was important in cases of this character, Dr. Hunt said, because of the treatment. The symptoms were growing progressively worse, and if they were the result of compression, osseous or otherwise, the advisability of an exploratory operation was worthy of consideration. This would be decided, the speaker said, on further observation and after an X-ray picture had been taken.

Diffuse Cauliflower-like Puckering of the Cortex in Arteriosclerotic Epilepsy, or Diffuse Cortical Cirrhosis.—Dr. Adolf Meyer demonstrated a brain that he had obtained through the courtesy of Dr. M. C. Ashley of the Middletown State Hospital. The specimen showed a form of vascular affection, principally of the cortical terminals, in a patient 55 years old. This patient gave a history of having had syphilis at the age of 20. There was mental deterioration for twelve years, and slowly progressive left hemiplegia during the last year of life.

The small vessels of the pia in the affected regions were diffusely occluded. The lesions affected principally the occipital lobes; the temporal and parieto-frontal regions were also affected, leaving, however, intact the convolutions bordering on the Sylvian fossa.

The case belonged to a group of which Dr. Meyer had described two instances in the Pathological Report of the Illinois Eastern Hospital for the Insane, at Kankakee, in 1896. The condition was frequently called diffuse sclerosis, but presented a distinct variety of a progressive disorder of middle life or senescence, akin to the cases reported by Pozzi, Hess, Prout, Blackburn, and probably also by Greiff. The term cirrhosis was used instead of sclerosis because the latter referred more to the broader lesions taking in the white substance, whereas cirrhosis, as applied to the kidney and liver, directed the attention more to the parenchymatous portions, and in this case to the cortex itself.

Dr. T. P. Prout said that in one case he had observed where the early history was obtained, it was found that it belonged to the realm of the infantile cerebral palsies, upon which epilepsy was subsequently ingrafted. This was the light, the speaker said, in which he had during recent years come to regard these cases. The epilepsy was ingrafted upon an early cerebral palsy in the same way as we saw it ingrafted upon other brain lesions.

Dr. Fraenkel said he had seen quite a number of cases in which epileptiform seizures developed late in life, accompanied by some mental deterioration. The diagnosis was of importance, because in a younger person such a condition would be regarded as a general paresis. It was interesting to study the onset of these seizures, which could usually be differentiated from those of ordinary epilepsy by the slight focal symptoms.

Dr. Meyer, in closing, said he did not think it was justifiable to assume an infantile lesion in these cases, because it was possible to demonstrate all degrees of more recent and older foci, with numerous granule cells. In reply to the suggestion made by one of the speakers to call these cases pseudo-paralysis, Dr. Meyer said this would be apt to involve them in the general confusion of that term, which for a while stood for the cases of general paralysis on a demonstrable syphilitic basis, but which we had since learned to recognize as the backbone of that disease.

The following officers were elected for the ensuing year: President, Dr. Joseph Fraenkel; First Vice-President, Dr. J. Arthur Booth; Second Vice-President, Dr. Smith Ely Jelliffe; Recording Secretary, Dr. J. Ramsay Hunt; Treasurer, Dr. G. M. Hammond; Corresponding Secretary, Dr. F. K. Hallock.

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 24, 1905.

The President, Dr. Charles S. Potts, in the Chair.

A Case of Cervical Hypertrophic Pachymeningitis.—This case was exhibited by Dr. William Pickett for Dr. L. C. Peter. Dr. Pickett said the woman had been attending the dispensary of the Medico-Chirurgical College for several years, most of the time under the care of the late Dr. Pearce. Her history is as follows: Four years ago she was confined to bed seven days with an acute illness, which was called peritonitis. Her husband had gonorrhea at that time. In the midst of her illness the woman complained of pain in the arms, back, shoulders and neck. Three months after this attack of peritonitis wasting of the hands was observed. At present there is wasting of the thenar and hypothenar eminences and of the other small muscles of the hand, with consequent flattening of the hand, producing the *main en griffe;* also symmetrical wasting of the forearms. The hands are exceedingly weak. Reactions of degeneration are present in these paralyzed parts. Knee-jerks are not altered. No ankle clonus and no Babinski are obtained. There is nothing remarkable in the case at present except the bilateral claw-hand. Evidently there was the painful or meningitic stage of the French writers, followed by the atrophic stage, but there is no supervention of the spastic stage. Dr. Pickett said that recently he had read the paper of Joffroy written in 1873, and it is remarkable how little has since been added to our knowledge of cervical hypertrophic pachymeningitis. It appears from Joffroy's paper that the disease was distinctly described, although not named, by Sir William Gull in 1858.

The apparent gonorrheal origin of this case was the point upon which Dr. Peter had laid stress.

Dr. Pickett said that this case was a legacy from Dr. Pearce's service at the Medico-Chirurgical Hospital, and that he (Dr. Pickett) knew only of the symptoms of the painful stage of the affection from the history and could not say how definite they were. He stated that he had seen one other similar case at Blockley, in which the sensory symptoms were very marked. From the history of the present case one would suppose they were not well marked. It seemed remarkable, too, that there is no involvement of the pyramidal tracts with increased knee-jerks, when the atrophy of the hands has reached such an advanced stage.

A Case of Epilepsy with Myoclonus.—This case was exhibited by Dr. Charles W. Burr. Dr. Burr said he first saw this boy in 1896. At that time he had a history of having had epileptic fits, beginning in the sixteenth month. At first they were very few. He had a series of fits at sixteen months, then an interval of twelve or fourteen months without another, then a few more convulsions and another period without any, and then convulsions occurred quite frequently. When Dr. Burr saw him in 1896 he had three or four attacks of minor epilepsy. He also had a pretty constant twitching of the muscles of the face and shoulders. It appeared then to be the kind of movement seen in habit spasm. Intelligence was good; the boy spoke well and had no palsies, no ataxia, no objective signs of disease except the apparent habit spasm of the head and shoulders, and he had a history of major epilepsy. Dr. Burr did not see him again until last April, when, on going into the ward at Blockley, he saw a boy in bed with violent choreiform movements, in severe chorea. He appeared to be having hallucinations, and told quite a story of having been assaulted by a physician who took him to a building, cut holes in him and sewed them up again. At that time Dr. Burr thought he had to do with a case of chorea with insanity. This diagnosis was incorrect, as 48 hours later he was sitting up perfectly quiet, had forgotten all about the story he had told, and to all appearances was a boy in fairly good health. A day or two after that he had an epileptic fit, which Dr. Burr saw. Since he has been in the hospital he has had eight or ten true epileptic fits. He has also had periods where for days he would have violent spasms of the voluntary muscles, including the muscles of the face; a violent myoclonic spasm lasting for hours or days. Sometimes this movement precedes a fit, sometimes follows one, and sometimes comes on independent of any fit at all. When the movements are bad voluntary movement makes it very much worse. If he attempts to walk he is often thrown down. Again, when the muscular spasm has been very great, for several hours afterward he will not walk. He is too weak to walk. He has no anesthesia, the reflexes are not abnormal, there is no disturbance of speech except the spasm of the mouth and lips, which causes stuttering. If he looks constantly to either side there is persistent oscillation of the eyeballs. At times he is stupid and wants to be let alone, but Dr. Burr had never seen him in any delirium. His skull is somewhat abnormally shaped, especially in the occiput.

Dr. Burr did not believe the case was one of hysteria, as hysteria does not begin at sixteen months of age, nor is it characterized by epileptic fits lasting for many years and showing the symptoms that this boy has. On the other hand, some of his attacks seem hysteroidal in character. He has never had hemianesthesia, reversal of fields of vision, nor anything of that kind. Dr. Burr said he had never seen a picture of disease exactly like this. It seemed to him to be best classified as a case of what Clark and Prout had called palsy with myoclonus. The patient's brothers and sisters were said to be perfectly sound.

Dr. Potts stated that this patient was under his care last summer, and he noticed that the movements grew worse, and indeed often commenced after a period of quiet, when any attention was paid to the boy; also that stern commands would frequently cause them to cease, which led him to believe that there was an element of hysteria in the case. While recognizing that he has epilepsy, Dr. Potts thought some of his symptoms could be attributed to hysteria.

Dr. Gordon had seen this case repeatedly, and stated that the jerky movements would often disappear for a time. The boy had often had falling attacks. He stated that at that time the history of epilepsy was unknown to him, and he thought it was a case of hysteria.

In closing the discussion on his case, Dr. Burr stated that there was concentric contraction of the visual field. He did not like to diagnose hysteria unless it was unavoidable, for it had been his misfortune to diagnose hysteria and afterward find organic disease. He stated that he had done this in cases where he thought there were hysterical convulsions, and which afterward proved to be epileptic convulsions. This boy had a hysterical manner and muscular spasm which varies from day to day, was sometimes controlled by effort of the will, sometimes controlled by voluntary effort, and sometimes made worse thereby. Still, with fits which are known to have been epileptic and a convulsive movement that is not hysterical, and as the case largely agrees with cases reported by Clark and others, Dr. Burr thought this to be a case of myoclonus epilepsy rather than a case of epilepsy plus hysteria.

A Case of Myasthenia Gravis with Paralysis Confined to the Ocular Muscles.—The patient was exhibited and the case was reported by Dr. William G. Spiller and Dr. E. U. Buckman. The case of myasthenia gravis was remarkable in that the weakness was observed only in the ocular muscles. The rapid exhaustion of the levator palpebræ superioris, first of one side and then of the other, especially when both eyes were uncovered at the same time; the recovery after rest, the variation in the paralysis of other external muscles of the oculomotor distribution, the integrity of the inner muscles of the eyeballs, a response of the sternocleidomastoid muscle to the faradic current, suggesting the myasthenic reaction, and the absence of all other signs of implication of the nervous system, make the case probably one of myasthenia gravis of the ocular type.

Notes by Dr. Spiller, Jan. 10, 1905.—The patient had severe headache during ten years, until about six or seven months ago. Vision was not affected when he had the headache, and has always been good.

When he takes off his glasses the upper lids begin almost immediately to droop and continue to do so until the eyeballs are covered, first one then the other. He wears a cover over one eye, and changes this from one glass to the other frequently. When he looks downward both upper lids fall. The external rectus on each side is normal. He cannot look upward or downward very well with the right eye, but can perform these movements with the left eye. He cannot keep both eyes open at the same time more than a minute, but if he covers one eye the other remains open. The iritic response to light and accommodation is prompt in each eye. Sensations for touch and pain are normal in all parts of the body. The muscles of the face, of the tongue and of mastication are normal. The biceps reflex is not unusually prompt on either side. The grasp of each nand is powerful. Voluntary power in the lower limbs is normal. The patellar reflex is normal on each side. Ankle clonus is not obtained. Gait and station are normal with eyes open or closed. The muscles of the facial distribution respond promptly to the faradic current.

Jan 24, 1905.—When he rises in the morning he cannot open his eyes so well as later in the day; formerly this was not the case. He can open his eyes better in the evening. During the entire day be uses his eyes in reading orders in his store (he is a groceryman) or in writing, therefore the disturbance is greater during the day, and is less in the evening, because he lies down.

When he takes off his glasses the upper lid of one eye, depending on which eye he had been using last, begins to fall, and gradually the upper lid falls until there is complete ptosis; while this is occurring the upper lid of the other eye gradually droops until ptosis may be complete or nearly complete on this side. The falling of the upper lid cannot be due to an attempt to overcome the diplopia, because the second lid droops after the first pupil is covered by the lid, and the falling of the lids occurs in the same manner when occasionally he has no diplopia while looking directly forward. He usually has diplopia in looking directly forward, but occasionally he has no diplopia for a few minutes, and then one eye "shoots off," as he expresses it, and diplopia develops. *When the upper lids have fallen, if he closes his eyes for two or three minutes he can open them again,* but with some ptosis of one lid; to-day it is in the left. He cannot keep both eyes open at the same time more than one or two minutes. If he covers the left eye with his hand the right eye stays open better, but after two or three minutes the right upper lid falls. He now can keep the left eye partially open if he covers the right eye. The weakness of the other ocular muscles seems now to be in the elevators and depressors of the right eyeball. The ocular palsy, he says, has seldom been the same at any two examinations. The deltoids react well to a rapidly interrupted faradic current, but there seems to be some exhaustion by this current in the left sternocleidomastoid muscle.

Dr. Sailer stated that he was reminded of a series of cases he saw two years ago. An old woman was brought to his office suffering with an entirely different condition, but she had partial ptosis of one eye and complete ptosis of the other. The ptosis became worse during any form of excitement. The daughter has had the same condition from early childhood. Whenever she became excited it became more pronounced. Her son, representing the third generation, had the same condition. After studying, or when fatigued, ptosis came on, more severe in one eye than the other, but always in both. No definite information could be obtained about her more remote ancestors, but it was supposed that her great-grandparent suffered from this peculiar form of transient ptosis. Two other children of the daughter are normal. Dr. Sailer did not test any case for the myasthenic reaction. None of the patients had ever complained of diplopia.

Dr. Gordon wished to put on record the case of a colored girl who presented a remarkable symptom group, which consisted of drooping of both eyelids. When he talked to her she would open the eyes, but the lids would immediately droop. She could put food in her mouth, but could go no further. Mastication and deglutition were very difficult. This condition persisted for several months, when the patient began to improve. In the case presented by Dr. Spiller and Dr. Buckman the eyelids drooped slowly, while in his case they drooped suddenly. Dr. Gordon regarded his case as one of profound asthenia of a certain group of muscles, probably a symptom of a curable case of myasthenia gravis.

Dr. Eshner thought Dr. Sailer's cases of ptosis were not of the myasthenic type. He mentioned a case of his own which had drooping of the eyelids persisting throughout the man's lifetime, but at that time nothing was known of myasthenia gravis. He also mentioned the case of a colored newsboy. The boy is often seen on the streets of Philadelphia and has ptosis, but probably not of the myasthenic type.

Dr. Mills stated that he had seen several cases of myasthenia gravis, but he had never seen a case of this kind. He mentioned the case of a woman past middle life, seen at the Polyclinic some years ago, who suffered from a peculiar form of drooping of the eyelids. The case was supposed to be hysterical. Hypnotic influence was tried and seemed to do good, but it has seemed to him since that if the case was myasthenic the improvement may have been due to keeping the eyelids closed for some time.

Dr. Spiller, in closing the discussion, stated that there is a condition of congenital ptosis, cases of which have been recorded, and it is possible that congenital ptosis can occur in females. He had seen hysterical ptosis, but thought in most of these cases the condition was one of hysterical spasm. He stated that he had considered the possibility of tumor in the region of the cerebral peduncles, of syphilis, of beginning tabes, of ophthalmoplegic migraine and of myasthenia gravis. He stated that the man was positive that he had not had syphilis. He had been given mercury and iodide in large doses. There is no evidence of tumor; tabes is not probable, as the knee-jerks are normal. There are no symptoms outside of the ocular paresis. Ophthalmoplegic migraine is unlikely because of Dr. Buckman's report on the case. The man is not hysterical and has no evidences of hysteria whatever.

Four cases of beri-beri were reported by Dr. Joseph McCool.

Dr. Pickett stated that he saw the last two of these cases with Dr. McCool, and that without the history of the cases he would have made a diagnosis of multiple neuritis of some other origin. However, dyspnea and tachycardia were still present when he saw the cases. He thought the most striking symptom was one observed in Dr. McCool's third case, namely, ataxia, which was quite marked. The man had to be supported, stepped wide, and put his feet down heel first as in peripheral pseudo-tabes. He had never heard a description of such marked ataxia in beri-beri.

Dr. Weisenburg stated that beri-beri is not a rare disease in the tropics. He had been in Manila when there were about one thousand cases distributed in the prisons. During his two-years' stay there he instituted a number of sanitary regulations which caused a great decrease in the number of cases. In the prisons of the interior there was no beri-beri, while in the prisons along the seacoast it was prevalent. The disease did not differ from neuritis except for the dropsical condition. There are three forms of the disease, the wet form, the dry form and the mixed form. They are all the same except in the amount of fluid present. He stated that there was a considerable degree of ataxia, and that it was rare for a patient suffering with beri-beri to be able to walk. As the patients improve, all show absence of knee-jerk and all become ataxic. In the large number of cases he saw in the Philippines he never saw a case among the soldiers.

The Pathology of Cerebellar Tumors was the title of a paper read by Dr. T. H. Weisenburg.

A Case of Spastic Ataxic Paraplegia Developing After Childbirth.—Dr. S. D. Ludlum reported this case at the December meeting (see p. 334) from the nervous dispensary of the Polyclinic Hospital. The patient, a woman, is married, aged 32 years. Her mother died of tuberculosis, her brother died of Bright's disease, but there was no nervous disease in the family. She had malaria and typhoid fever at the age of 14. She was married at 20 years of age, and has four children living and well, and has had no miscarriage.

Twelve years ago she had her first child. After this for about one month, she says, she had her eyes crossed and had vomiting spells. She then wore glasses, and in three months seemed to be well. The second child was born and she had no disorder following. The third child was born six years ago. When she attempted to get out of bed she found that her left lower limb "gave way" and felt numb, but was not painful to pressure. The general health was good. The weakness and numbness lasted one year and then disappeared. Two years and six months following the birth of this child the fourth child was born, three years ago. She then had a "melancholy spell" of four or five months' duration; at the same time, as soon as she got out of bed she found that there was weakness of both hands (not the arms) and a numb feeling. The left leg was weak. The weakness in the arms disappeared, but continued in the left leg, and about Jan. 1, 1904, both lower limbs became affected. She had no pain, headache nor vertigo, and could hold the urine fairly well and had no rectal disturbance. She was treated at the Woman's Medical Dispensary for pelvic conditions, and was later admitted to the University Hospital, where a blood examination showed a considerable grade of anemia with spastic gait, both legs dragging, toes scraping, and some ataxia, more marked with eyes closed. The patellar reflex much exaggerated on each side. The Achilles tendon reflex was exaggerated on the left side, and lessened on the right side. She had a slight indication of ankle clonus on the right side, but not on the left. Babinski's reflex was distinct on each side, and she had a slight tendency to talipes equino varus. Sensations of touch and pain were slightly diminished in the lower limbs, and she had also subjective sensation of cold in these limbs. The upper limbs were normal, except for slight exaggeration of the reflexes. The cranial nerves were normal.

The eyes were examined by Dr. Shumway. The left nerve had a pathological pallor, the right was of better color, both eyes having refractive error.

She left the hospital somewhat improved, and has since been coming to the Polyclinic Hospital, and has continued to improve on iron and arsenic and tonic treatment, until about Nov. 15, when she says she saw double and was unable to read or thread a needle. Her vision would be good for a short space of time and then return to the abnormal condition.

The eyes were examined by Dr. Posey in December. Paralysis of right inferior rectus and paretic condition of the left inferior rectus and oblique muscles were found. The pupils reacted on both sides, but there was a paralytic condition of accommodation. Both nerves showed pallor on the temporal side, this perhaps indicating a commencing simple atrophy. At present she has very little trouble with her eyes, except that when reading she has a feeling of slight strain.

Strümpell, in a recent article in the *Deutsche Zeitschrift für Nervenheilkunde,* Vol. 27, in writing of spastic spinal paralysis, speaks of the possibility of a beginning systemic spinal disease in women after pregnancy and childbirth. He has seen unusual cases of spastic spinal paralysis which had a relation to childbirth, but as he had no anatomical data, he calls the attention of other observers to these cases.

The possibility of this case presented by Dr. Ludlum being one of multiple sclerosis must be considered, on account of the pallor of the temporal side of the optic disks and the temporary ocular palsies first appearing twelve years ago.

Starr has reported a case of multiple sclerosis developing after childbirth, and Balint has reported a case in which, after four successive confinements, a rapid increase in the symptoms followed.

The long duration of the disease without the development of typical symptoms would make one cautious in diagnosing multiple sclerosis. In the *Lancet* in July, however, are given a number of cases illustrating the frequent occurrence of remissions or recoveries or relapses as a feature of multiple sclerosis.

Müller, in going over nineteen atypical cases, mentions four without either nystagmus, scanning speech or intention tremor.